Digestive tract

DIAGNOSTIC APPROACH TO COLIC IN ADULT HORSES

- PAIN – degree, duration, and type
- PULSE – rate and character
- PERFUSION – mucous membranes, skin tent, jugular fill, etc.
- PERISTALSIS – gut sounds, fecal production
- PINGS – simultaneous auscultation/percussion
- PASSING A TUBE – amount and character of reflux, if present
- PALPATION – rectal exam
- PAUNCH – a word for obvious abdominal distention that begins with “P”
- PCV/TP
- PERITONEAL FLUID

MEDICAL MANAGEMENT OF COLIC IN ADULT HORSES

- ***Non-steroidal anti-inflammatory drugs (NSAIDs)-*** Flunixin meglumine, ketoprofen, and phenylbutazone are non-selective inhibitors of COX1 and 2, whereas carprofen and etodolac are somewhat COX-2 selective.
- ***α-2 adrenergic agonists -*** xylazine, romifidine and detomidine, can provide excellent sedation, analgesia, and muscle relaxation for horses with severe abdominal pain.
- ***Opiods:*** **butorphanol**
- ***Anti-spasmodics:*** **N-butylscopolammonium bromide has both anticholinergic and antispasmodic properties**

- ***Decompression -*** gastric decompression via a nasogastric tube, cecal enterocentesis in the right paralumbar fossa
- ***Alternate analgesia:*** **Intravenous lidocaine has been used in horses both as an analgesic and as a treatment/preventative for post-operative ileus.**
- ***Oral fluids -*** 6-8 L every 4-6 hours **Always check for reflux prior to administration, and never administer enteral fluids to a horse with more than 1-2 liters of reflux.**
- ***Intravenous fluids:*** half of the calculated fluid deficit within the first 1-2 hours, with replacement of the remaining deficit (plus maintenance and ongoing losses) over the next 12-24 hours.

- **LAXATIVES –**

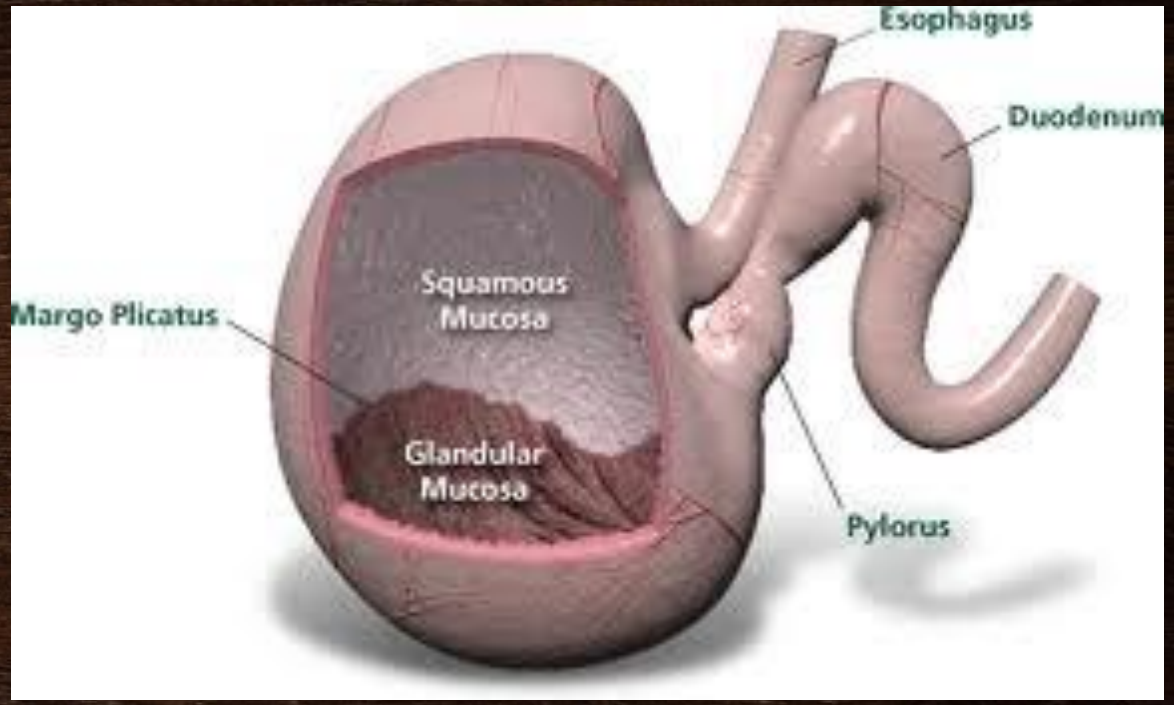
✔ mineral oil at 0.5-1 gallon via NGT in an adult horse.

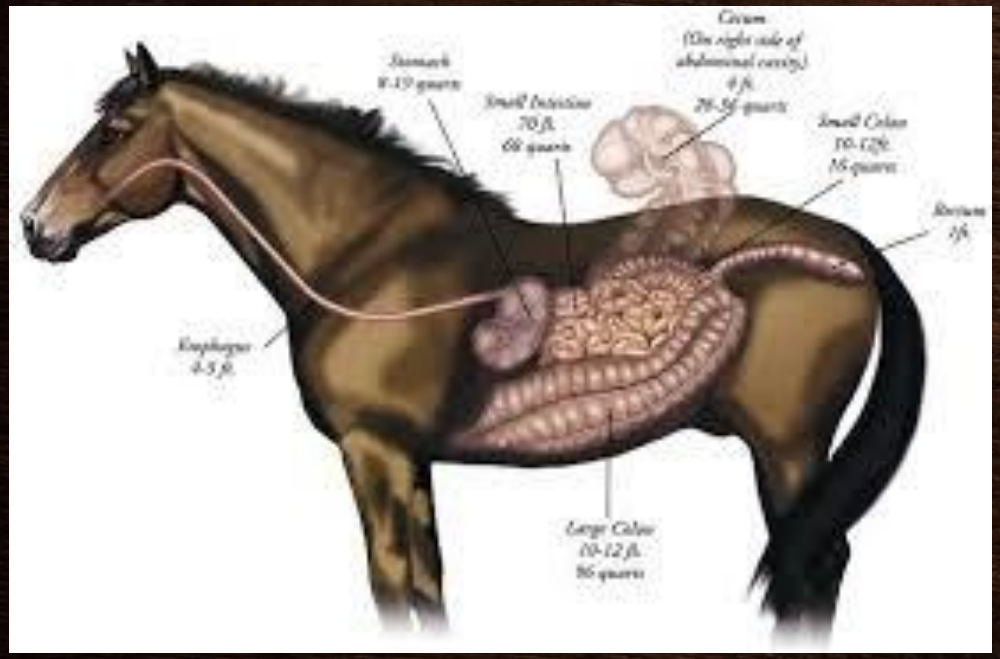
✔ magnesium sulfate (Epsom salts; 0.5-1 g/kg in 8 L water)

✔ Psyllium mucilloid

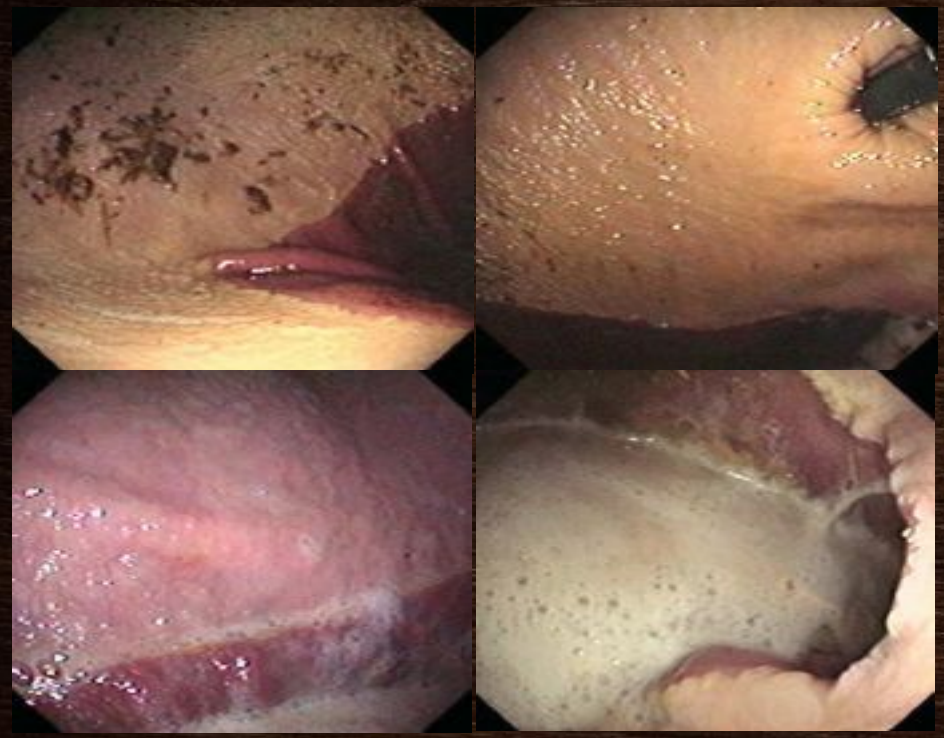
stomach

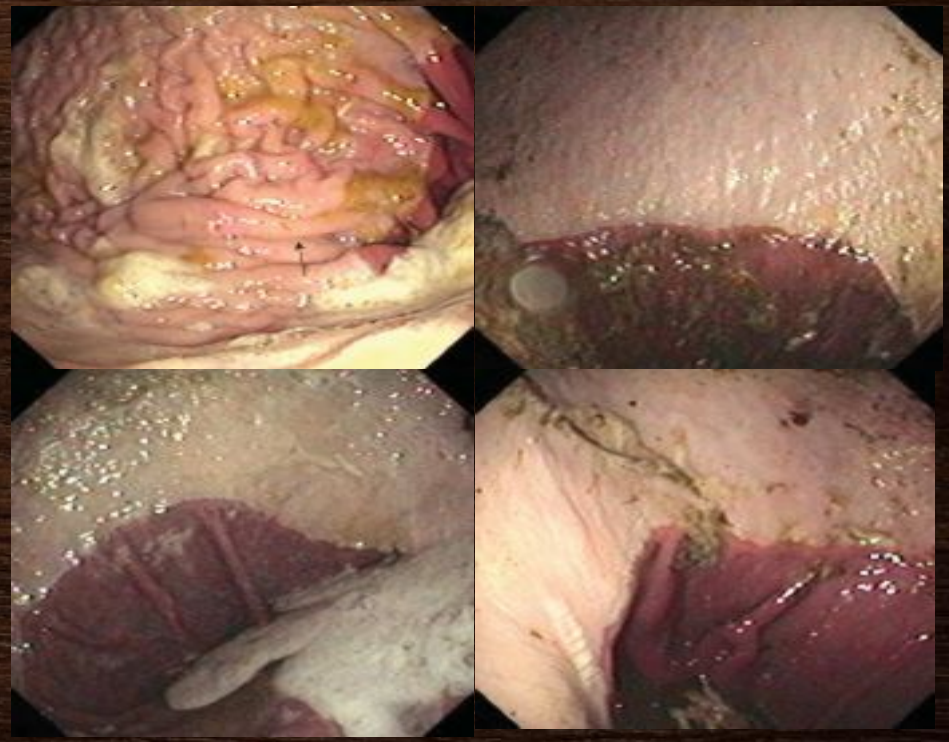
- The horse's stomach is relatively small, with a capacity of approximately 10 to 15 liters, and is situated dorsocranially in the left side of the abdomen within the confines of the ribcage.
- The oblique nature of the cardia renders it virtually impossible for the horse to vomit and thus makes passage of a nasogastric tube a critical component of the diagnostic work up of horses with colic.


Esophagus
Duodenum
Squamous Mucosa
Margo Plicatus
Glandular Mucosa
Pylorus


Cecum
(On right side of abdominal cavity)
4 ft.
28-36 quarts
Stomach
8-10 quarts
Small Intestine
70 ft.
68 quarts
Small Colon
10-12ft.
16 quarts
Rectum
1ft.
Esophagus
4-5 ft.
Large Colon
10-12 ft.
86 quarts

Stomach





DO PREZENTACJI UŻYTO ZDJĘĆ Z ATLAS OF EQUINE ENDOSCOPY N.M.SLOVIS WYD MOSBY

Gastritis

- Gastritis is an inflammation and irritation of the lining of the stomach.
- Unlike a stomach ulcer, gastritis involves large areas. The mucosa--inner lining --throughout much of the stomach appears red and swollen, and contains many small ulcerations or areas of erosion

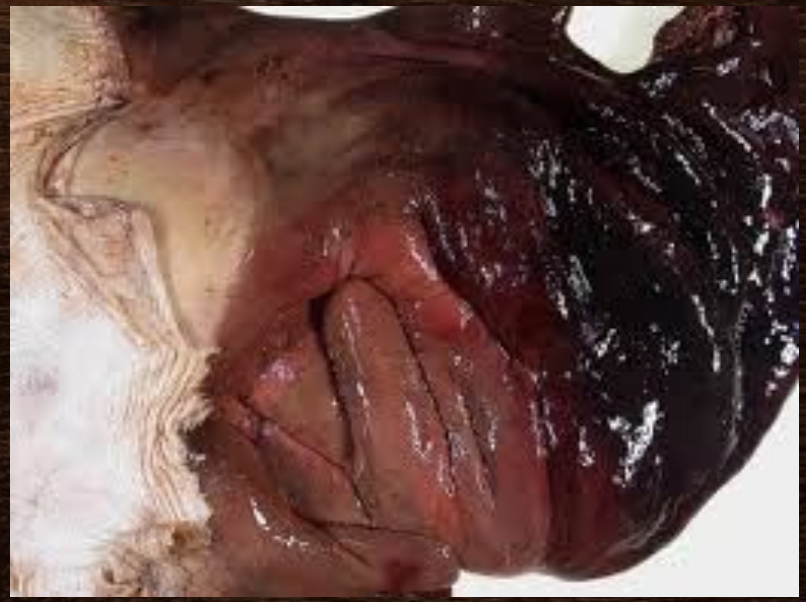
Acute Gastritis

- Acute gastritis is caused by ingesting moldy or spoiled feed, sand, chemicals and toxins, or by overeating.
- Laminitis--a metabolic and vascular disease which involves the inner sensitive structures of the feet--can accompany or follow an episode of acute gastritis.

Chronic Gastritis

- Chronic gastritis is associated with the long-standing ingestion of poor quality feeds or foreign materials such as wood shavings, sand, or stones. These ingestible materials irritate the lining of the stomach and often remain for long periods, during which they combine with feed to form bezoars--impacted feed balls. The bezors are too large to pass into the small intestines but are small enough to intermittently block the outlet of the stomach.

- The horse with acute gastritis salivates, vomits, and drools excessively, refuses to eat and exhibits colic.
- Signs of chronic gastritis include intermittent colic, lack of appetite, weight loss and bad breath



treatment

- H2 receptors blockers – ranitidin 6-7 mg/kg every 8 hours, cymetidin 10-20 mg/kg
- Proton pump inhibitors – omeprazol 2-4 mg/kg
- Good hey , pasturage

Gastric ulcers

- Ulcers are a common medical condition in horses and foals. It is estimated that almost 50% of foals and 1/3 of adult horses confined in stalls may have mild ulcers.
- Up to 60% of show horses and 90% of racehorses may develop moderate to severe ulcers.
- Because they are so common, and can occur as a result of a number of factors, the condition is often called "equine gastric ulcer syndrome" (EGUS) or "equine gastric ulcer disease" (EGUD).

- Stomach is divided into two distinct parts. The non-glandular portion (also called the esophageal region) is lined by tissue similar to the lining of the esophagus. The glandular portion is lined with glandular tissue, which produces hydrochloric acid and pepsin, an enzyme needed for the digestion of food.
- In the horse, however, hydrochloric acid is constantly being produced. So, if a horse does not eat, the acid accumulates in the stomach, and can start to irritate the stomach, especially the non-glandular portion.

Causes of gastric ulcers

- Fasting (not eating) - Horses evolved to graze, eating many small meals frequently. This way, the stomach is rarely empty and the stomach acid has less of a damaging effect. If horses and foals do not eat frequently, the acid builds up and ulcers are more likely to develop.
- Type of feed - The type and amount of roughage play a role in ulcer development. Roughage, because it requires more chewing, stimulates the production of more saliva. The swallowed saliva helps to neutralize stomach acid. There is an increase in acid production when concentrates are fed. The type of roughage is also important. Alfalfa is higher in calcium, and it is thought that this may help decrease the risk of ulcers.
- Amount of exercise - As the amount of exercise increases, there is often a change in feeding (e.g., more times of fasting, less roughage), which increases the risk of ulcer development. In addition, exercise may increase the time it takes for the stomach to empty, so large amounts of acid can remain in an empty stomach for a prolonged period of time. Stress itself may decrease the amount of blood flow to the stomach, which makes the lining of the stomach more vulnerable to injury from stomach acid.
- Medications - Chronic use of non-steroidal anti-inflammatory drugs (NSAIDS) such as phenylbutazone (Bute) and flunixin meglumine (Banamine) blocks the production of a particular chemical called PgE2. PgE2 decreases acid production, so when PgE2 levels are low, acid levels are high, contributing to the development of ulcers.

Signs of gastric ulcers in horses

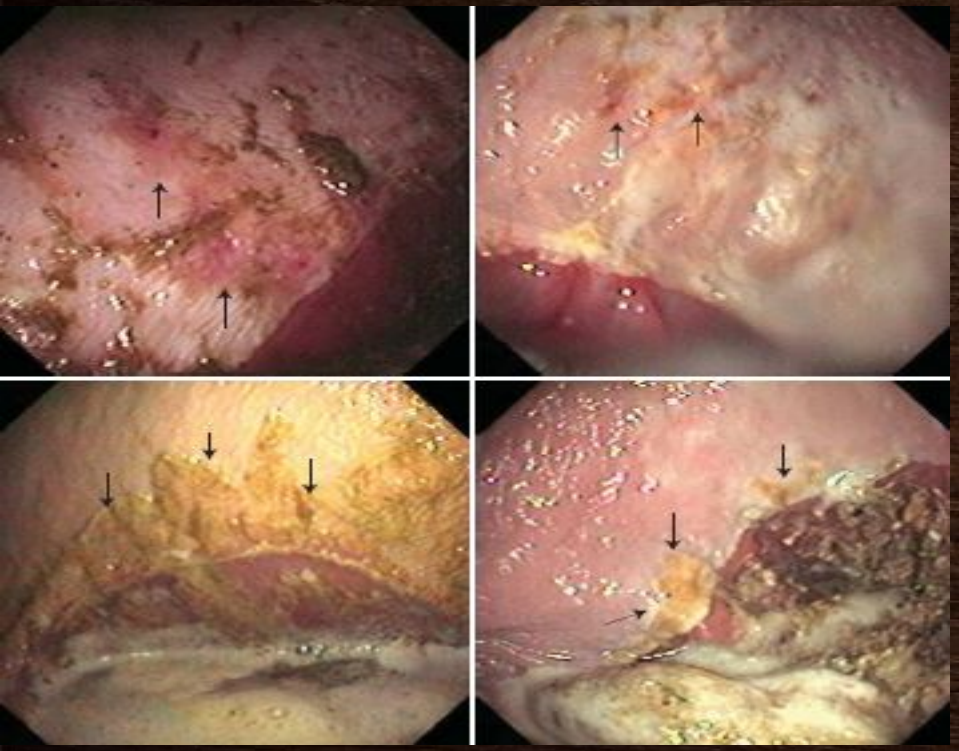
In foals, signs of gastric ulcers include:

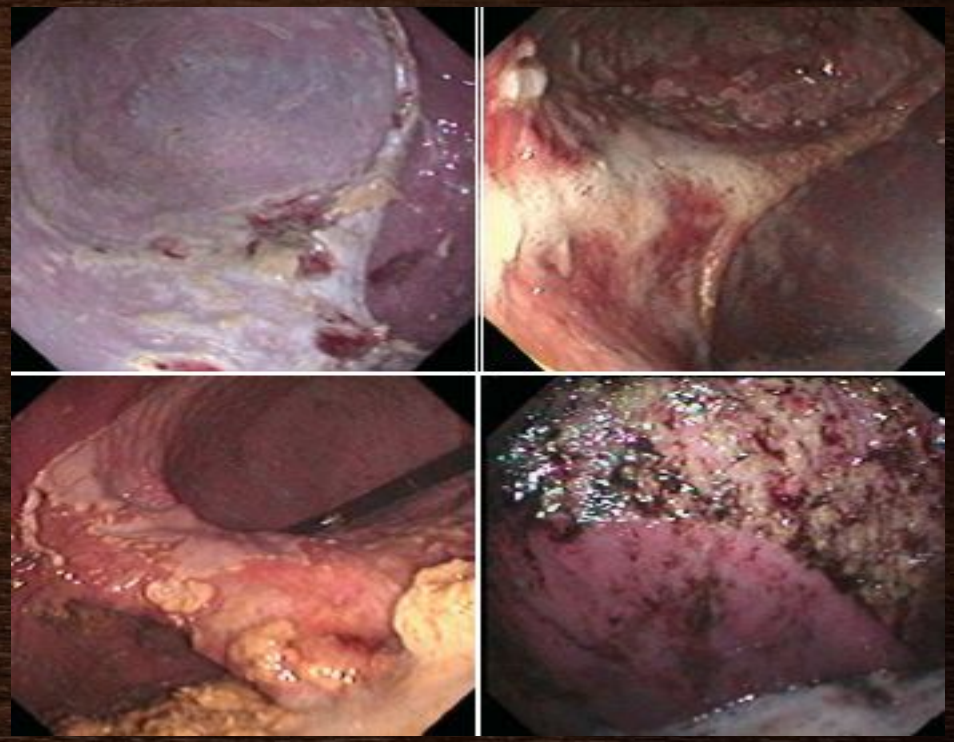
- Intermittent colic, often after nursing or eating
- Poor appetite and nursing for only very short periods
- Teeth grinding
- Excessive salivation
- Diarrhea
- Lying on the back

In adult horses, signs of gastric ulcers include:

- Poor appetite
- Weight loss and poor body condition
- Poor hair coat
- Mild colic
- Mental dullness or attitude changes
- Poor performance
- Lying down more than normal

Stomach ulceration





III grade

DO PREZENTACJI UŻYTO ZDJĘĆ Z ATLAS OF EQUINE ENDOSCOPY N.M.SLOVIS WYD MOSBY

Treatment of gastric ulcers in horses

- H2 blockers: These are medications that block the action of histamine. Histamine stimulates the production of stomach acid. Example: Cimetidine, ranitidine.
- Proton pump inhibitors: These are medications that inhibit the production of acid by the stomach
- Buffers: Antacids buffer the action of the stomach acid. Because acid is constantly being produced in the horse, antacids are effective for only a short time (less than an hour) and require large amounts be given. This makes them relatively impractical in the horse, though their use on the day of performance or a stressful event may be beneficial.
- Protectants: Certain drugs can block acid from coming into contact with the stomach lining. Unfortunately, these do not appear to be as effective in the esophageal portion of the stomach. Example: Sucralfate.

In addition to medications, changes in management are almost always necessary including:

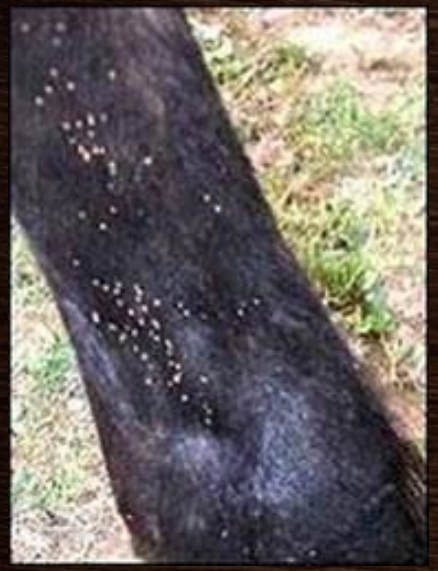
- Increasing the amount of roughage in the diet.
- Increasing the number of feedings by increasing the amount of time the horse is actually eating. Putting the horse on pasture would be the best alternative.
- Avoiding or decreasing the amount of grain. Use supplements to add the vitamins and minerals, and vegetable oils to add the calories the horse may need.
- Giving probiotics to aid in digestion.

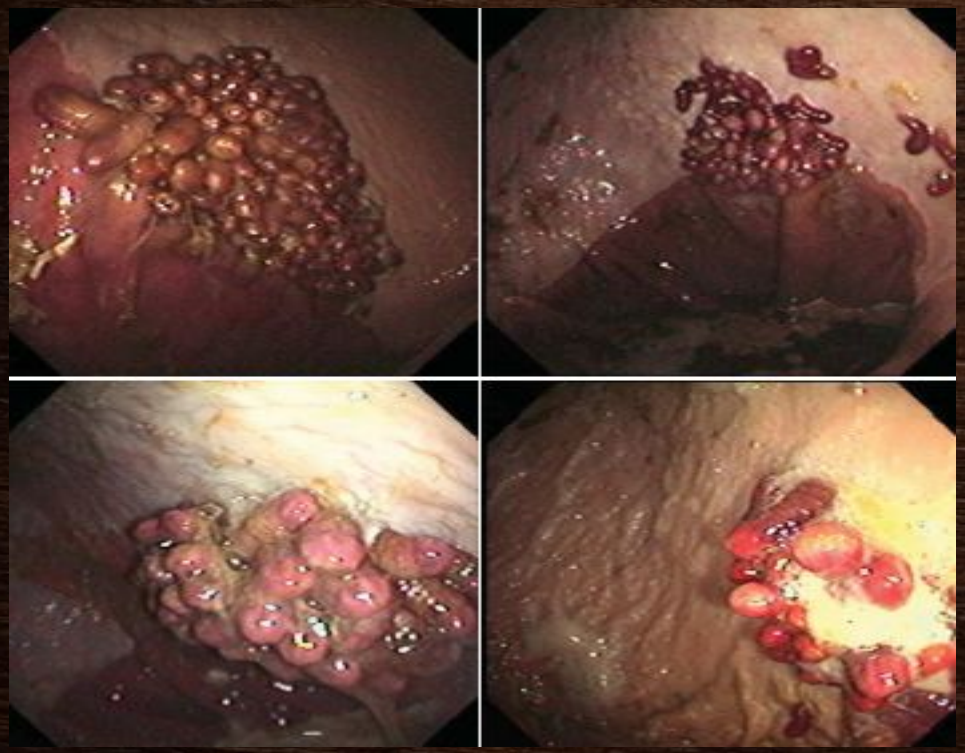
Gastric parasites

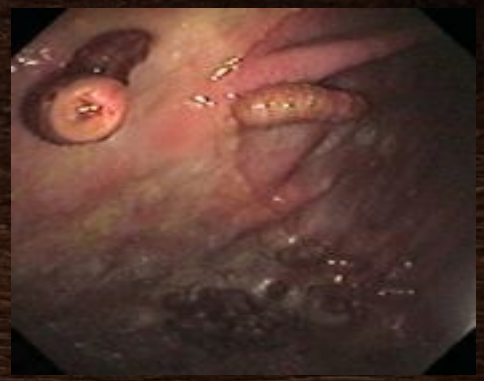
- Horse bots, which are found in the stomach, are the larvae of botflies, Gasterophilus spp.
- The eggs of *G intestinalis* (the common bot) are glued to the hairs of almost any part of the body but especially the forelimbs and shoulders. The eggs of *G haemorrhoidalis* (the nose or lip bot) are attached to the hairs of the lips. *G nasalis* (the throat bot) deposits eggs on the hairs of the submaxillary region.

- The larvae of all 3 species apparently stay embedded in the tongue or the mucosa of the mouth for ~1 mo, after which they pass to the stomach where they attach themselves to the cardiac or pyloric portions
- The main pathogenic effect is caused by larvae, which attach by oral hooks to the lining of the stomach. This induces erosions and ulcerations at the site of attachment and a hyperplastic reaction around it.

- Bots cause a mild gastritis, but large numbers may be present with no clinical signs. The first instars migrating in the mouth can cause stomatitis and may produce pain on eating. The adult flies may annoy horses when they lay their eggs.
- Ivermectin is effective against oral and gastric stages of bots







ATLAS OF EQUINE ENDOSCOPY N.M.SLOVIS WYD MOSBY

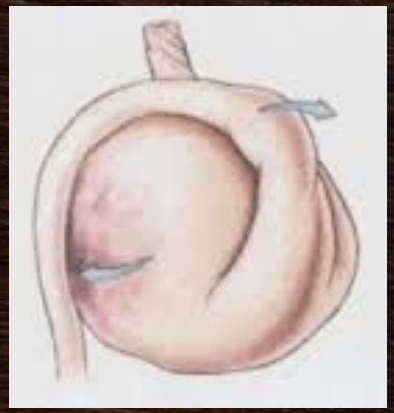
Gastric dilatation and rupture

- Gastric dilatation can be classified as primary, secondary, or idiopathic.
- Causes of primary gastric dilatation include gastric impaction, grain engorgement, excessive water intake after exercise, aerophagia, and parasitism.
- Excessive consumption of fermentable feeds (grains, lush grass, and beet pulp) causes a large increase in the production of volatile fatty acids which is thought to delay gastric emptying.

- Secondary gastric dilatation occurs more commonly and can result from primary intestinal ileus or small or large intestinal obstruction. Fluid from the obstructed small intestine accumulates in the stomach, causing nasogastric reflux. Gastric dilation may also occur with certain colonic displacements, especially right dorsal displacement of the colon around the caecum. Gastric fluid accumulation is also characteristic of proximal enteritis-jejunitis.
- Time to development of reflux is proportional to the distance to the segment involved, with duodenal obstruction resulting in reflux within 4 hours.

Gastric dilation usually produces:

- Acute, severe colic
- Tachycardia
- Pale mucous membranes
- Retching
- Ingesta at the nares in severe cases (rare)
- Gastric reflux



Gastric rupture typically results in:

- Relief
- Depression

The inevitable peritonitis and endotoxic shock will lead to:

- Reluctance to move
- Tachypnoea
- Tachycardia
- Sweating
- Muscle fasciculations
- Blue or purple mucous membranes

Primary gastric dilation should be suspected :

- copious amounts of gastric reflux in the absence of small intestinal distension on rectal examination and the absence of endotoxaemia.
- colic signs cease following decompression, and other clinical parameters return to normal.
- does not cause any significant change in peritoneal fluid parameters until rupture occurs.

Secondary gastric dilation should be considered:

- persistent colic, repeated retrieval of nasogastric reflux, intestinal distension on rectal examination and clinical signs of endotoxaemia.
- indications for exploratory laparotomy to look for an intestinal obstruction.

Gastric rupture results in septic peritonitis which will be reflected in the nature of fluid collected by abdominocentesis:

- Foetid, turbid sample containing particulate matter
- White cell count >40 x 109/l
- Protein content >30g/l.

Findings on rectal examination may include:

- A 'gritty feeling' on the serosal surfaces of intestine due to adherent food material
- An impression of 'space' in the abdomen due to gas in the peritoneal cavity.

Laboratory findings may include:

- Haemoconcentration
- Hypokalaemia
- Hypochloraemia

treatment

- Gastric lavage (water or oil)
- Treat underlying disease

Gastric Impaction (Obstruction)

- Gastric impaction can result in either acute or chronic signs of colic.
- Although a specific cause is not always evident, ingestion of coarse roughage (straw bedding, poor quality forage), foreign objects (rubber fencing material), and feed that may swell after ingestion or improper mastication (persimmon seeds, mesquite beans, wheat, barley, sugar beet pulp) have been implicated.
- Possible predisposing factors include poor dentition, poor mastication and rapid consumption of feedstuffs, and inadequate water consumption.

Clinical signs

The colic associated with gastric impaction varies from mild and chronic to acute and severe.Other signs reported include:

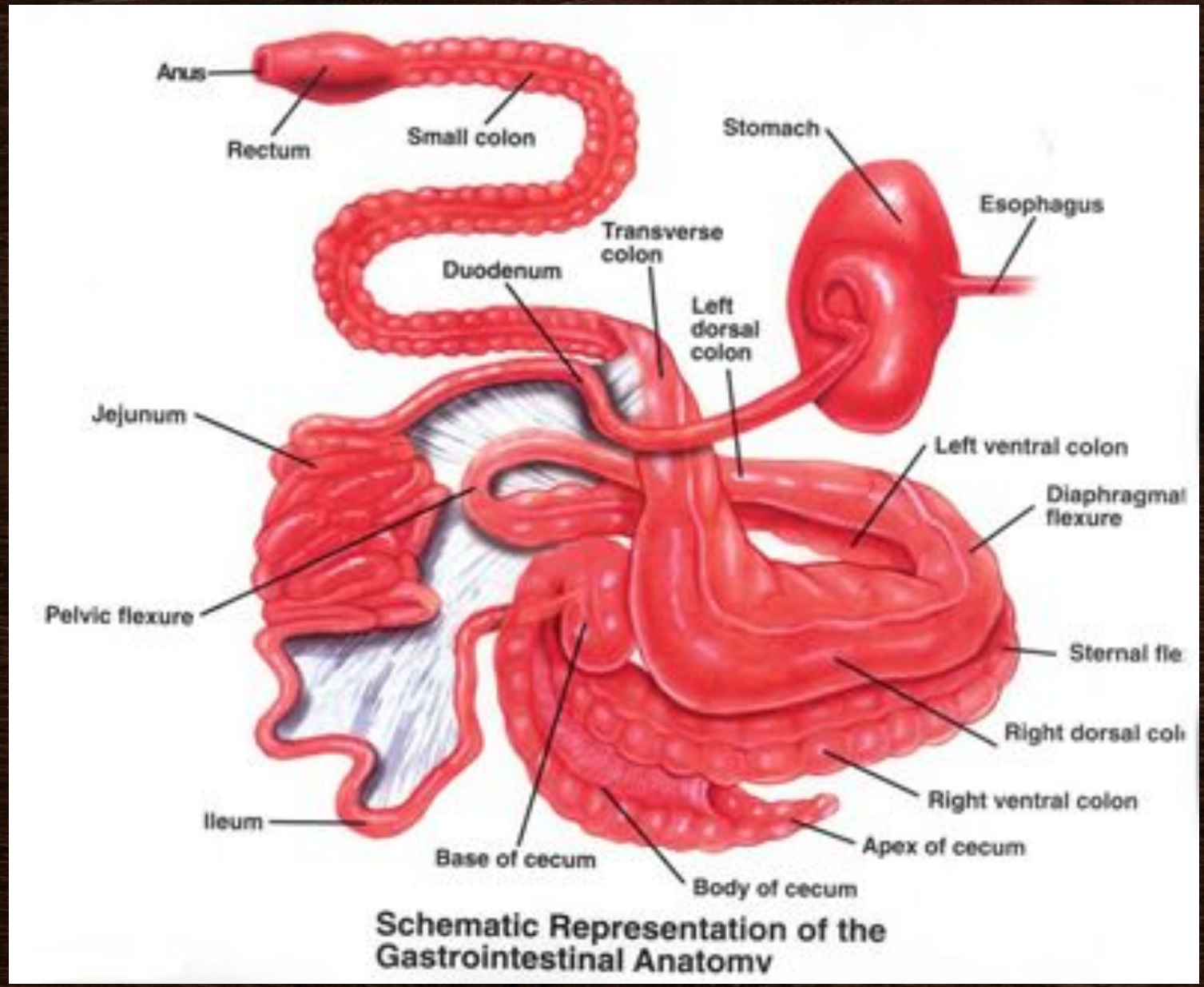
- Anorexia
- Lethargy
- Prolonged recumbency
- Dysphagia
- Dropping of feed
- Bruxism
- Salivation
- Insidious weight loss (if chronic)
- Spontaneous reflux with gastric contents visible at the nares (in severe cases)

treatment

- gastric lavage with water
- IV fluid therapy and analgesia
- the impacted stomach can be felt extending back midway between the xiphisternum and the umbilicus
- Infusion of balanced polyionic fluids such as saline either directly into the impaction through the gastric wall (adjacent to the greater curvature) or via a nasogastric tube
- Massage of the stomach to reduce the impaction and aid movement of fluid into the ingesta
- Impactions diagnosed at surgery may benefit from bethanechol to stimulate gastric motility.
- The stomach should be lavaged by nasogastric tube post-operatively and the horse starved for 48-72 hours.

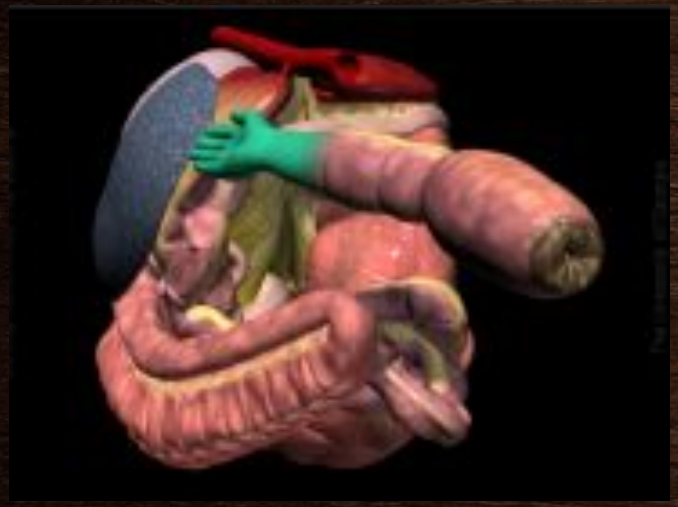
prevention

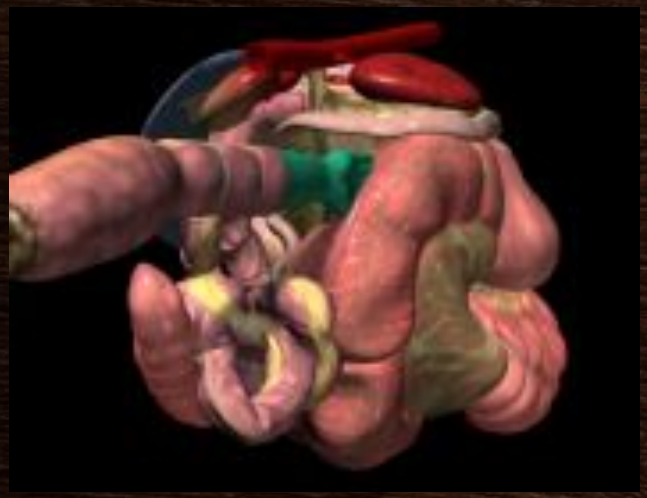
- Regular dental care
- Ensure sugar beet nuts are adequately soaked prior to feeding
- Secure storage of roughage and hard feeds
- Ensure free access to water at all times
- Good pasture management to prevent ragwort poisoning

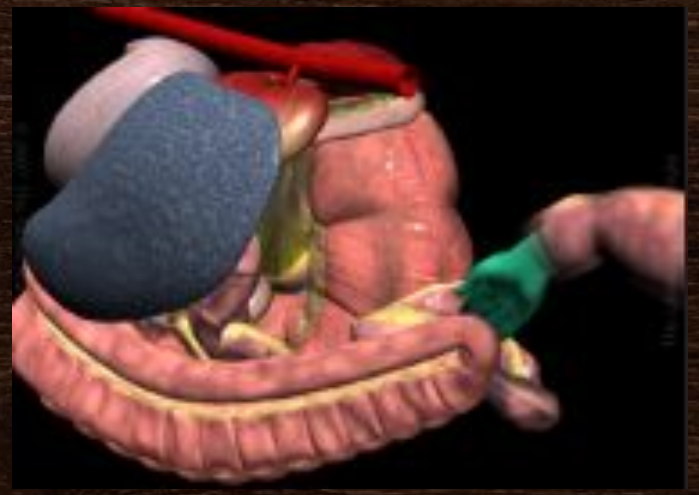


Schematic Representation of the Gastrointestinal Anatomy

Rectal examination

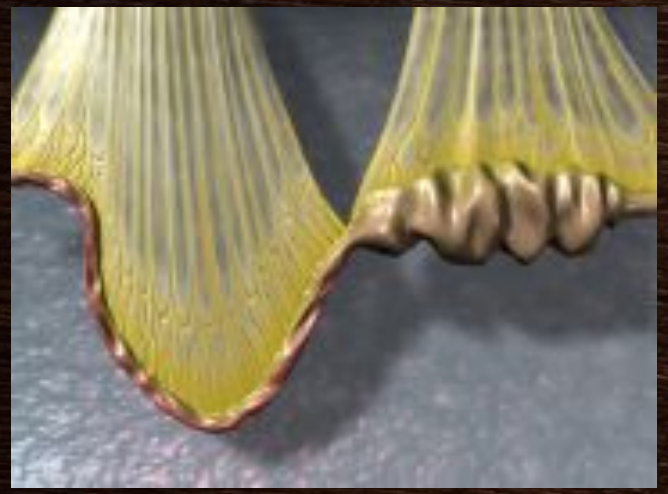


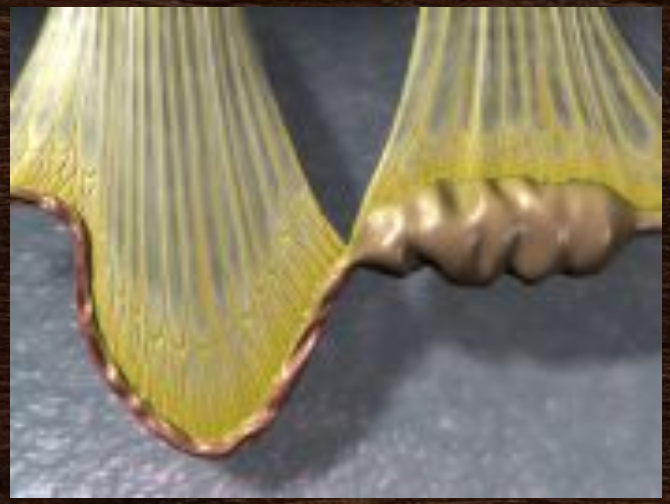




Obstruction

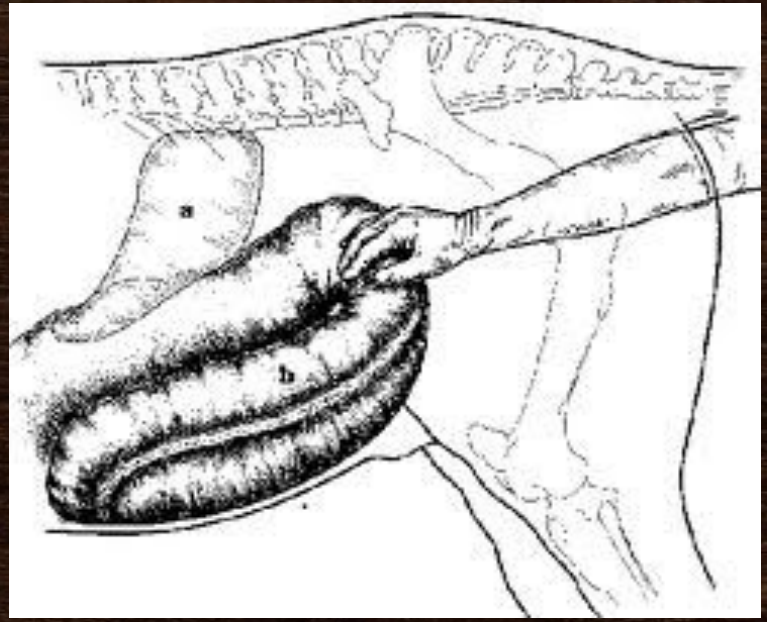
- normal movement of ingesta is restricted or prevented but no change occurs in the blood supply to the intestine
- occur when ingesta fails to move from a portion of the bowel having a large diameter into a portion with a smaller diameter
- impaction of the large colon at the pelvic flexure, enterolithiasis,





Pelvic Flexure Impaction

- occur when dry or inadequately digested feed fails to move through the **pelvic flexure, the region connecting the large left ventral colon with the** smaller left dorsal colon
- additional ingesta fills the entire **left ventral colon**



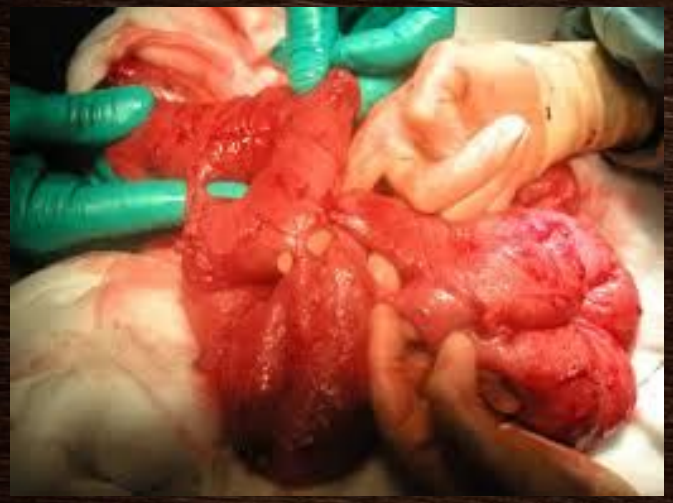
Signs and treatment

- **mild abdominal pain**
- heart rate slightly increased
- intestinal sounds usually can be heard

- oral and intravenous administration of fluids
- mild analgesics
- laxatives

Adhesions

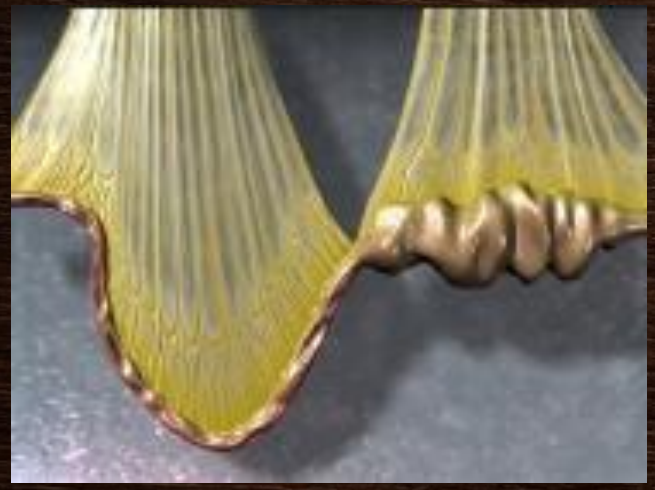
- develop as a complication of previous **small intestinal surgery** or because of parasite migration, abdominal abscesses,penetrating abdominal wounds, or serosal inflammation

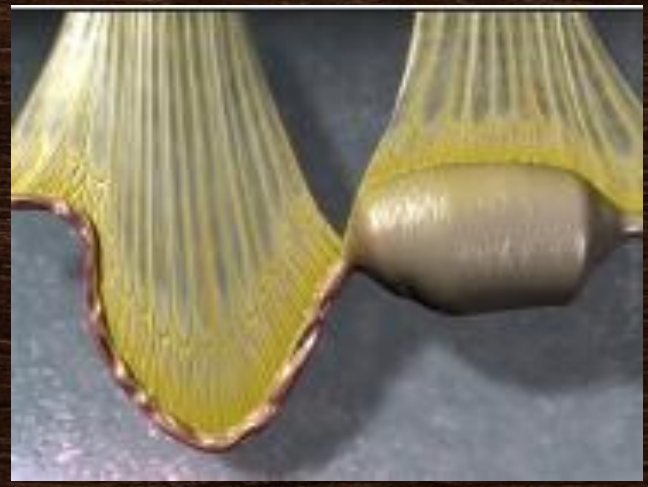


- history of a gradual onset of colic and weight loss, and in many instances the pain occurs after the horse eats
- diet to facilitate movement of ingesta, or, more often,
- surgery to remove the affected segments of intestine

Distention

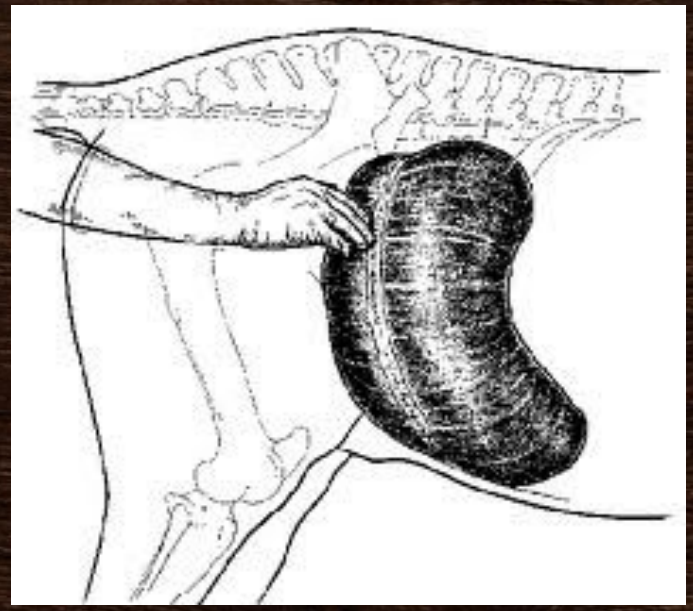
- occurs when excess gas in the intestinal lumen stretches the wall of the intestine
- cecal tympany and gastric dilation





Cecal Tympany

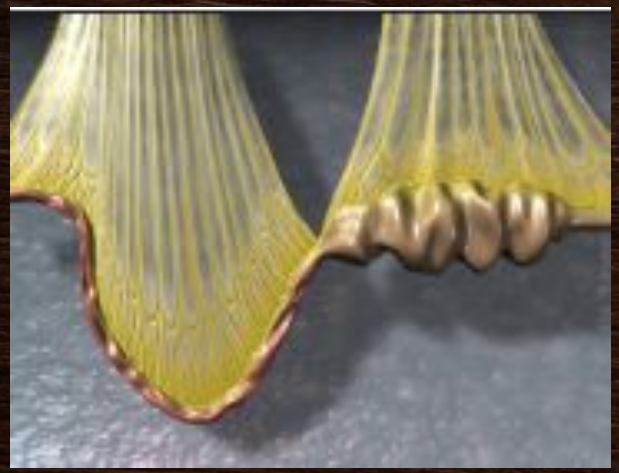
- occurs commonly in horses with colonic displacements, colon volvulus, or obstruction of the small colon
- as a primary disease due to the rapid fermentation of lush pasture grasses or an abrupt change in diet

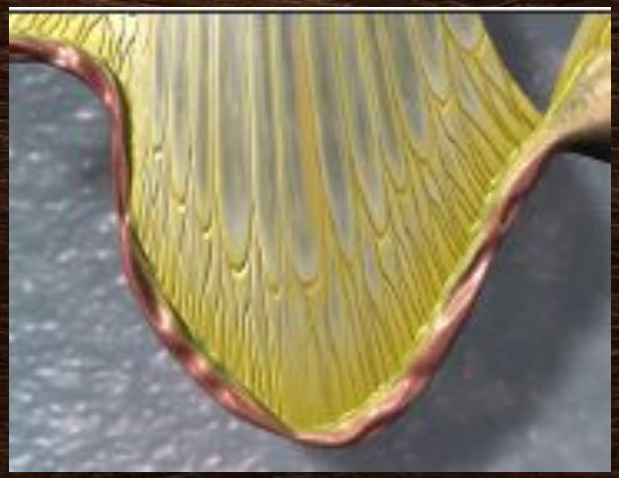


- distention of the abdomen
- tight paralumbar fossae
- pain
- tachycardia and tachypnea
- high-pitched pinging sound in the right
- removal of the gas through a trocar

Spasm

- abnormal, uncoordinated contractions of smooth muscle cells in the wall of the intestine
- the blood supply to the intestine is normal, and there is no obstruction to the movement of ingesta



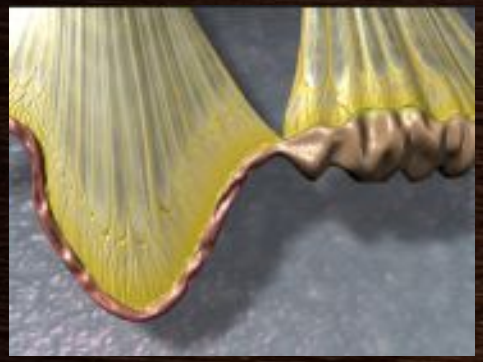


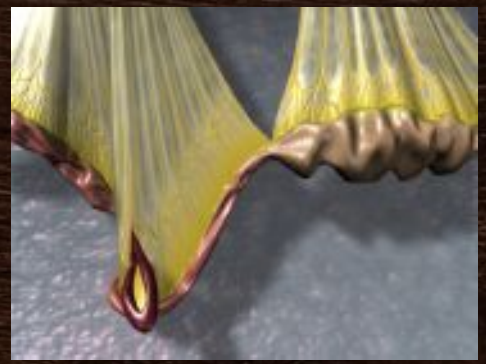
Spasmodic Colic

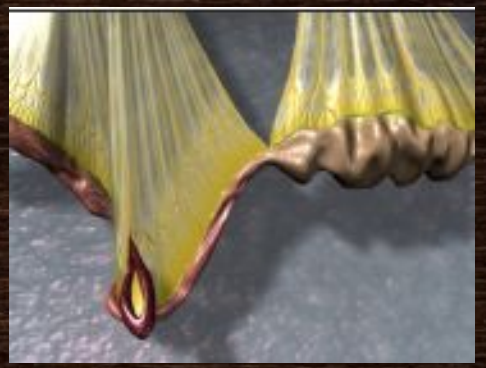
- occurs due to **spasm or cramping of intestinal musculature**
- diagnosis is based on the **lack of other findings**
- abdominal pain is relieved by administration of mild analgesics or spasmolytic agents

Strangulation Obstruction

- occur when both the flow of ingesta and the intestinal blood supply are interrupted
- occur if the intestine moves through an opening, such as a tear in the mesentery, or if the intestine twists enough to occlude the lumen and the vessels
- large colon volvulus, inguinal hernia, and incarceration of small intestine through a mesenteric rent

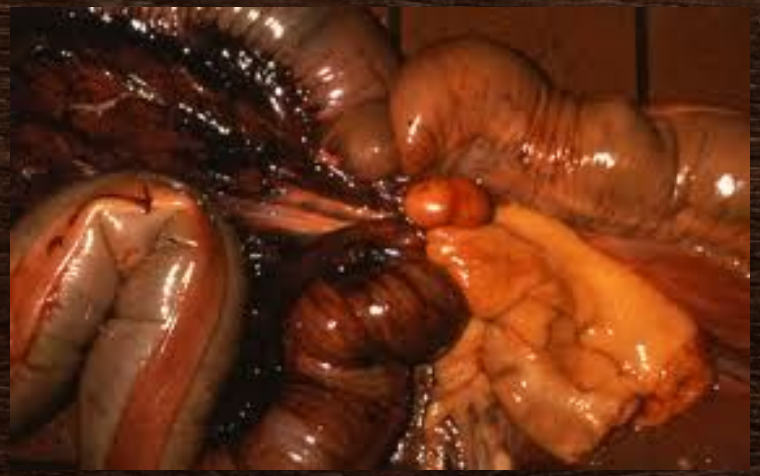






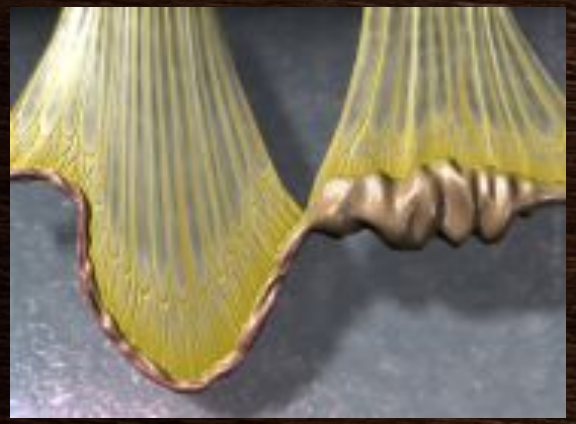
Small Intestinal Strangulation through Mesenteric Rent

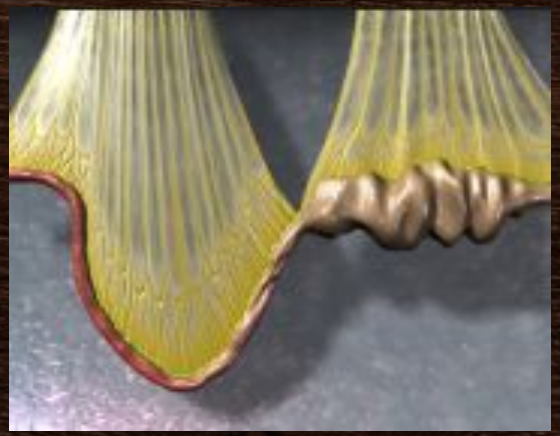
- defect in the small intestinal mesentery called a **mesenteric rent**
- a loop of the jejunum to pass through a mesenteric rent
- outflow of blood and lymph from the intestinal loop is impeded
- horses are painful, toxemic, hemoconcentrated, and dehydrated

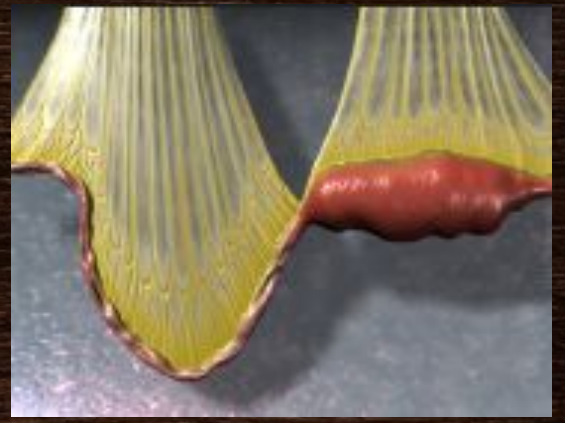


Enteritis and Colitis

- Enteritis refers to inflammation of the small intestine. This inflammation results in thickening of the intestinal wall, secretion of fluid into the intestinal lumen, and distention of the intestine with gas and fluid
- Colitis refers to inflammation of the colon. The inflamed colonic wall becomes edematous, and large volumes of fluid are secreted into the colonic lumen

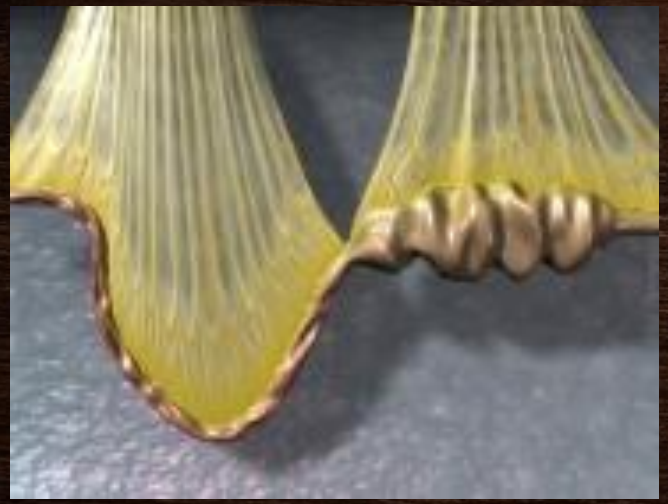


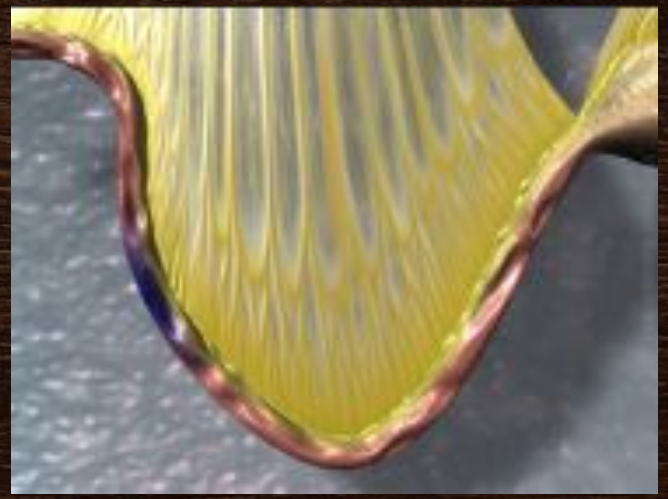




Nonstrangulating Infarction

- Loss of blood supply to part of the intestine in the absence of a displacement or incarceration





- **thromboembolism or** a **reduction in local blood flow**
- secondary to parasitism
- postoperative

Signs:

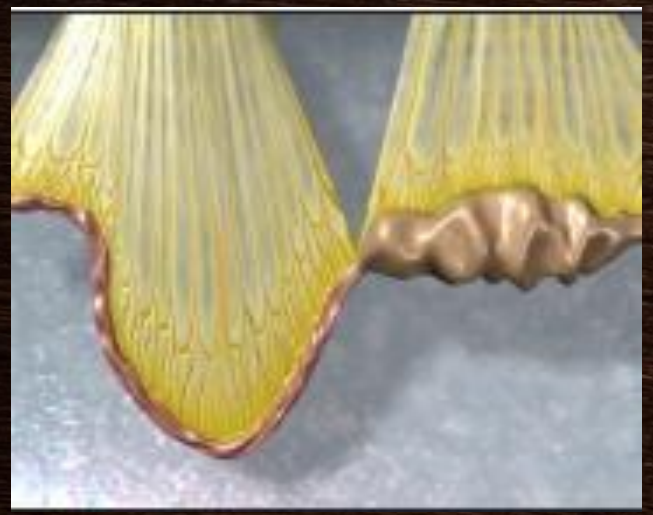
- chronic intermittent episodes of mild to moderate abdominal pain
- deterioration of the systemic circulation
- depresion
- when complete infarction of the intestine
- Very strong pain and distended colon

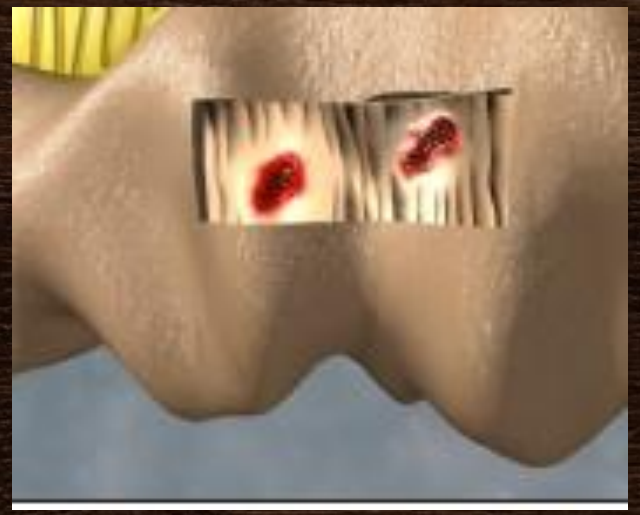
Treatment:

- analgesics
- intravenous fluid replacement
- larvicidal
- aspirine/heparine

Ulceration

- Loss of mucosal epithelial cells
- may result in bleeding into the intestinal lumen and even perforation of the intestinal wall
- gastric ulcer disease, which occurs in the stomach, and right dorsal colitis, which occurs in the right dorsal colon





Peritonitis

- occurs secondary to strangulated or severely inflamed intestine and results in the movement of large numbers of white blood cells into the peritoneal cavity